

CRITICISM OF THE SEPTEMBER 15, 2006 SAB DRAFT ADVISORY REPORT AS IT APPLIES TO INORGANIC ARSENIC

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There are two major assumptive flaws in the analysis of inorganic arsenic health data as presented by EPA Offices and reviewed by the SAB Review Panel: (1) animal data extrapolation, which is used to supplant rather than explain actual human health findings; and (2) the extrapolation of high dose human cancer data to low dose, which is used to supplant significant, contrary actual low dose human findings. [These flaws particularly influence the responses the SAB Review Panel crafts to Charge Questions B3, C2 and D2.] Since the Review Panel approved the use of the Taiwan epidemiology data set, which has ample bladder (and lung and liver) cancer data in the exposure range of interest (10-60 $\mu\text{g/L}$) to generate significant findings, there is no defensible reason to rely on extrapolations from other species or from higher arsenic doses in Taiwan villagers.

The failure of the SAB Review Panel to challenge these assumptions results in its wasting time and effort on asking for the development of uncertainty analyses tied to the extrapolations, which generate incorrect default-driven conclusions. On Pages 48-50, the Review Panel fails to critically examine the data-driven notion separately published on by Lamm and Kayajanian that from 0- or 10-60 $\mu\text{g/L}$, the bladder cancer death rate has a significant negative slope, deriding Lamm's work, not even referencing Kayajanian's. If, as the Review Panel claims on page 49, lines 27-29, "[t]here is (sic) no human data available that is (sic) adequate to characterize the shape of the dose response curve below a given point of departure..." how can Lamm and Kayajanian find and publish on them? [Emphasis added.]

The actual bladder cancer data in the Taiwan data set EPA relied on to justify lowering the drinking water standard from 50 to 10 $\mu\text{g/L}$, actually shows a significant three-to-four fold increase in villages with arsenic levels at 10-32 $\mu\text{g/L}$ compared to villages with arsenic levels "around 50 $\mu\text{g/L}$ " (i.e., 42-60 $\mu\text{g/L}$). In the cover letter to the Administrator [page 2, lines 22-25], the Review Panel misleads: "... the dose response for human data in the low dose region does not describe clearly the shape of the curve, but they do fit with a linear model." What these low dose data do fit are a linear model with a negative slope.

This low-dose anti-carcinogenic response applies to the other cancers in the Taiwan data set (lung and liver) and to other cancers in other studies, especially the Millard, Utah data set that EPA scientists collected and published on but EPA regulators ignored. [After all, isn't an endpoint of lung plus liver plus bladder, or total cancer mortality more relevant than bladder cancer alone?] Inorganic arsenic at high exposures may be carcinogenic, compared to arsenic at 50 or 0 $\mu\text{g/L}$, and carcinogenic responses at high exposures may be explained by the fancy pharmacokinetic and animal studies the Review Panel discusses – but these explanations are inappropriate for arsenic exposures below 50 $\mu\text{g/L}$, which is where EPA has been regulating arsenic levels.

By failing to notice the significant cancer reductions associated with arsenic “around 50 µg/L” compared to 10 or less than 10 µg/L, the Review Panel has failed to fully and responsibly respond to the important anti-carcinogenic data finding that is finessed by several Charge Questions. The Review Panel’s Report, generally supporting the Agency’s analysis, should be rejected as inadequate and harmful (cancer-wise) to the American population impacted both by the current 10 µg/L arsenic-in-drinking-water standard and the expected further reduction of the arsenic standard below a 10 µg/L level. A successor Review Panel with total new membership, unburdened by this Panel’s failures, should address the questions the EPA Offices asked and chose not to ask this SAB Expert Panel.
